Cerebral Evoked Potentials in Schizophrenia

COMPUTER ANALYSIS OF electroencephalographic (EEG) responses to repeated stimuli yields information not ordinarily available in the usual clinical EEG. Thus, such evoked potentials (EPS) are used to test simple sensory competence (for example, EP audiometry). However, EPS also reflect more subtle cognitive processes. Schizophrenia is characterized by a variability of cognitive functioning as manifested, for example, by disorderly thought processes. This excessive cognitive variability is reflected in an excessive EP variability. High EP variability is no more unique to schizophrenia than is cognitive variability—both are also found in children and in Korsakoff's psychosis. Cognitive instability can usually be assessed adequately by interview. However, when language fails—as for example, because of cultural differences, language barriers or mendacity, EP variability can provide evidence on cognitive function.

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Narcolepsy and Cataplexy—Innovations in Diagnosis

THE PRIMARY PATHOLOGICAL process in narcolepsy is the attack of sleep. Just as there are two kinds of normal sleep, rapid eye movement (REM) and nonrapid eye movement (NREM), so do there appear to be two kinds of narcolepsy. In one, the abnormal process is sudden overwhelming attacks of REM sleep, and in the other, the abnormality is attacks of NREM sleep. It is difficult, on the basis of history alone, to distinguish between the two. However, if one or all of the auxiliary symptoms of cataplexy, sleep paralysis, and hypnagogic hallucinations are present, the presumptive diagnosis would be REM-type narcolepsy. It is important to confirm this impression with a polygraphic recording which necessarily includes electro-oculogram, and electromyogram properly recorded, in addition to the EEG.

An attack of REM sleep or a sleep onset REM period associated with the characteristic EMG suppression, rapid eye movements, and saw-tooth waves in the EEG, in other words, a period of REM sleep exactly like that occurring normally at night, establishes the diagnosis. There are a number of dissociative processes in REM-type narcolepsy. The physiological components which ordinarily occur together in normal REM periods may occur in isolation, thus, the motor inhibitory component of REM sleep in isolation is cataplexy. The dream component of REM sleep in isolation is the hypnagogic hallucination. In the Stanford University Sleep Disorders Clinic we have seen only two patients who had the presumptive diagnosis of NREM narcolepsy. In one case, the diagnosis was changed when sleep onset REM periods developed following withdrawal from amphetamines. In the other case, the sleep attacks disappeared following withdrawal from methylphenidate. It is our opinion that a number of instances of NREM narcolepsy are in reality a drug dependency hypersomnia. Thus, REM narcolepsy may eventually develop into NREM narcolepsy due to the development of dependency on these stimulating drugs, plus their tendency to suppress REM sleep.

Treatment of REM-type narcolepsy should begin conservatively with attention to nocturnal sleep and daytime naps, if possible, and use of methylphenidate and amphetamines. Other drugs which act primarily on the motor inhibitory process are the tricyclic antidepressants, and the monoamine oxidase inhibitors. Nardil® appears to be the most easily tolerated of the latter. A combination of tricyclic antidepressants with a regimen of naps will sometimes control narcolepsy so that amphetamines do not have to be employed.

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